



Original Communication

Alumnium phosphide fatalities at mild exertion in asymptomatic children: A clue to understand the variations of the autopsy findings

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ABSTRACT

Fatalities resulted from aluminium phosphide (ALP) intoxication in completely healthy children with no preceded clinical signs or symptoms were presented. Data regarding circumstances, autopsy reports, histopathological examination, toxicological investigation, and police enquiries were also collected and evaluated. The affected children were females, and 6–16 years old. They were completely healthy and died suddenly in relation to some physical activities such as running, walking, and bathing, without any prior complain. The viscera showed intense congestion with moderate to severe pulmonary edema. The cause of the sudden termination of life in the reported cases is mostly cardiac ones. Physical exertion may precipitate death due to increased cardiac stress, increased oxygen demand, and by aggravating metabolic acidosis. The absence of clinical symptoms before death may be due to the low level ALP, or due to the occurrence of death in the early stages after exposure to poison. Death due to ALP could result in cases of mild, moderate, or severe ALP intoxication. This may explain partly the differences encountered in clinical, autopsy, and histopathology findings of ALP intoxication. Fatalities are not the mere consequences of the dose of the poison, but factors such as physical activity and low oxygen level may be quite important.

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1. Introduction

Aluminium phosphide (ALP) is widely used all over the world as a fumigant to protect stored grain from pests and rodents.^{1–3} It is marketed in Jordan as 3 g tablets under several brand names as Celphos, Phostek, Quickphos, and Gastoxin.¹ It is a highly toxic compound that releases phosphine gas on contact with moist surfaces.⁴

In some countries, ALP is often implicated in accidental and suicidal poisonings.^{4–7} Reported poisoning in children was mostly accidental. Ingestion and inhalation of the poison were responsible for most of the fatalities from ALP.^{1, 7–9}

The findings in such fatalities showed no difference between children and adult cases.

Macroscopic examination during the autopsy in its case revealed one or a combination of major visceral congestion, pulmonary edema, and petechial haemorrhages on the surface of liver and brain.^{20,11,12} As for the microscopic examinations in various organs of the body, they are more variable and may show one or more of the following changes: For the lungs, those changes may be encountered, alveolar thickening, edema, dilated capillaries, collapsed alveoli, haemorrhage, cellular infiltration, and gray/red hepatization. As for the liver, patchy areas of necrosis in the heart, central

venous congestion, degeneration, haemorrhage, sinusoidal dilation, bile stasis, centrilobular necrosis, Kupffer cell hyperplasia, nuclear fragmentation, vacuolization of hepatocytes, infiltration by mononuclear cells, and fatty changes may be encountered. Degeneration, infiltration, tubular dilation, and cloudy swelling of the kidney, congestion, and coagulative necrosis in the brain, congestion, and haemorrhage in the stomach, and complete lipid depletion, haemorrhage, and necrosis in the adreno-cortex may appear.^{10–15}

On the other hand, there were few reported cases regarding inhalation of the poison. The findings vary from congestion, pulmonary edema with atelectatic areas in the lungs, focal myocardial infiltration, to small-vessel injury.^{8,16–18}

Aluminium phosphide when ingested or inhaled is highly toxic. The occurrence of death and the variation in the autopsy findings is mainly related to the dose of the poison.^{2,4,17,19}

This study is aimed to present five sudden fatalities resulted from ALP intoxication in completely healthy children with no preceded clinical signs or symptoms. This will highlight the high vulnerability of children to the poison, and will also help in understanding the discrepancies of autopsy findings on exposure to ALP.

2. Materials and methods

Sudden unexpected fatalities in children who have not complained of any preceded clinical signs or symptoms, and where

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the cause of death was attributed to ALP were studied. Data regarding the circumstances, autopsy reports, histopathological examination, toxicological investigation, and police enquiries were collected and evaluated. Toxicological investigations were conducted at the police forensic laboratories. Liver and blood samples were taken from the victims, and blank and spiked specimens were analyzed for ALP by liberation of phosphine gas from the specimen for qualitative detection.²⁰ The method was based on successive reactions of phosphine gas with silver nitrate, chlorine gas, ammonium molybdate, and benzidine.²⁰

3. Results

There were five proven fatalities that have resulted from ALP poisoning. All of them were females, and their ages varied between 6 and 16 years old. In their past history, they were described to be in good general condition. They were completely healthy and had no complaint what so ever especially in the past few hours preceding their deaths, provided that their deaths was as a surprise to their families. They were exposed to ALP accidentally. Their exposure to ALP was in the vicinity of grain stores (grain elevators and groceries), or inside their homes, and occurred within the spring season (March and April).

3.1. Circumstances of death

All of the cases died suddenly in relation to some physical activities without any prior complaint. A 6-year-old girl dropped suddenly to the ground in the morning of a celebration day (Eid), while she was running. She then raised her head once or twice when she stopped moving and died. Grain groceries were located in the nearby region where empty ALP containers were not infrequently found. Two other sisters 6 and 8 years age were awoken early in the morning having their breakfast and then started their journey to school by walking. They were living about 400 m away from the school. At noon, and in their way back to their home, both of them dropped suddenly to the ground. Their death nearly occurred in a period of 5 min apart. A grain elevator and groceries were located in their way where empty ALP containers were frequently found. The last two cases were identical twins of 16 years old. They had their bath in the afternoon. One hour later and outside the bathroom and after finishing their bath, both of them fell to the ground dead. Their death was separated by about 5–10 min. ALP tablets were sometimes used in their bathroom to kill cockroaches and pests.

3.2. Autopsy findings

External examination of the body revealed marked lividity of normal distribution with frothy and bloody exudates coming out of the nose and mouth. No injuries could be detected except for the two sisters, where superficial abrasions and bruises in the forehead, chin, and knees were found. Internally, the viscera showed intense congestion with moderate to severe pulmonary edema and frothy blood tinged exudates in both lungs. The stomach contained semi liquid materials in all cases with some solid ones especially in the two sisters where the contents were greenish to blackish in color too. The cause of death was attributed to acute heart failure and pulmonary edema.

3.3. Histopathology investigation

Microscopic examination disclosed minimal changes in the form of edema and congestion in most body organs. The lung's tissue showed variable degrees of alveolar and interstitial edema.

Sub-endocardial flame haemorrhages was especially found in the heart of the two sisters. Minimal neutrophil inflammatory cells could be seen in the cardiac muscle of the 6-year-old child.

3.4. Toxicology analysis

Toxicological analysis revealed presence of phosphine in blood and liver specimens of all cases. Drug screens were negative. Elevated carboxy hemoglobin (5%) was recorded in both twins.

4. Discussion

Prove of exposure to ALP depends on the history, postmortem examination, and laboratory investigation. Phosphine gas as evidence of ALP exposure was found in all victims, utilizing the procedure described by Curry.²⁰ It is concluded that the route of exposure was by ingestion in the two sisters and by inhalation in the other cases.

Immediate death, due to the exposure to ALP, results from pulmonary edema, while delayed deaths are presumably related to a direct cardiotoxicity.^{17,21–24} The deaths could be related to dose of poison consumed, severity of poisoning, duration of shock, failure of response of shock to resuscitative measures, and severe hypomagnesaemia.^{2,4,10,19}

Mortality from ALP poisoning showed great variation in the time taken by the poison to cause death, clinical presentation, autopsy, and histopathology findings.^{11,21,25–27} The presence of these differences points out to the presence of different mechanisms of death due to ALP. In the presented cases of this study, sudden death of children was precipitated by mild to moderate exertion in the form of walking, running, and bathing.

Many studies point out indirectly to the vulnerability of children to ALP. In one report, two deaths were reported in fumigated boxcars.²⁸ Four males, ages 12, 35, 39, and 52 years, were discovered in a boxcar containing loose bulk lima beans that had been fumigated with aluminium phosphide. The men had been in the car for approximately 16 h and had periodically opened the hatch for fresh air as needed. When discovered, the 12-year-old was dead and the other three men were asleep, suggesting that children may be more susceptible than adults. The three survivors reported nausea, vomiting, headache, and abdominal discomfort.

In another report, Aluminium phosphide fumigation aboard a grain freighter resulted in an acute illness in two female children and 29 of 31 crew members.¹⁶ One of the two exposed children died (age 2 years). The surviving child (age 4 years, 9 months) exhibited nausea, vomiting, dizziness, epigastric pain, and fatigue. The electrocardiogram, ECG revealed tachycardia with depression of the isoelectric period (ST segment), and an echocardiogram 24 h later showed dilation and poor function of the left ventricle. A transient increase in the cardiospecific isoenzyme fraction of serum creatinine phosphokinase CPK-MB was also observed indicating evidence of myocardial injury. Clinical signs and symptoms were resolved within 18 h, and ECG, echo, and CPK abnormalities were resolved within 72 h. Crew members exhibited shortness of breath, cough, sputum production, nasal drainage, nausea, jaundice, vomiting, diarrhea, fatigue, headache, drowsiness, paresthesias, tremor, and weakness.^{16,28}

Although this is the first time to report ALP fatalities initiated by exertion, another study may indirectly support this presentation. Approximately 200 chickens were found dead, due to zinc phosphide exposure, after the flooring of a slat-and-litter house was breached. No clinical signs of illness were observed in the surviving birds.²⁹

In general, most exercise-related sudden deaths in children and young individuals are cardiac deaths.³⁰ Accordingly, the cause of

sudden termination of life in the reported cases is mostly a cardiac one. ALP is known to affect the heart by causing toxic myocarditis and disturbances in magnesium ions,^{23,31} in addition to its production of generalized hypoxia and metabolic acidosis in the tissues.^{3,14,19,26,32,33}

Physical exertion may precipitate death in these cases due to increased cardiac stress and increased oxygen demand and by aggravating the metabolic acidosis. The role of the relative decrease in oxygen supply in precipitating death is seen clearly in the twin case with a carboxy hemoglobin level of 5%.

The absence of clinical signs and symptoms before death in the reported cases may be due to the low level of ALP, or to the occurrence of death in the early stages after exposure to poison. This is supported by other studies that found that initial signs and symptoms of intoxication from phosphine gas may be mild or nonspecific and transient.^{34–36}

Due to the sudden type of death, the mere fall to the ground may be insufficient to cause bruises in cases of the two sisters. This type of injury is mostly precipitated by the haematological effects of phosphine poisoning. Although the haematologic system is not a major target in phosphine/phosphide poisoning, intravascular hemolysis, reduced blood coagulation, reduction of platelets, and purpura were reported in non-fatal cases.^{37–41} Moreover, in fatal cases widespread, small vessel injury and small haemorrhages were also found postmortem.^{42,43}

The suggested precipitation of death by mild and moderate physical exertion points in a way or another that other factors other than the dose of ALP, may play a crucial role in causing death in exposed cases. Accordingly, death may occur at different levels of ALP toxicity. This may explain partly the differences encountered in clinical, autopsy, and histopathology findings of ALP fatalities. Moreover, it can be concluded that death due to ALP could result in cases of mild, moderate, or severe ALP intoxication.

5. Conclusion

This study points clearly that fatalities resulted from ALP are not the mere consequences of the dose of the poison, but other factors such as physical activity and low oxygen level may be quite important. Accordingly, Environmental Protection Agencies interested in setting regulations regarding registration and use of phosphine fumigants should take into consideration the difficulty in defining safe levels of exposure, especially for children.

Conflict of interest statement

None declared.

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Ethical approval

No ethical approval is needed.

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